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EFFECTS OF HYPOXIA ON PERIPHERAL VISUAL RESPONSE TO
DIM STIMULI

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occurring within 90 minutes followed by gradual recovery. Since the present results showed less impairment than previous data for brighter stimuli using the same task, it is concluded that stimulus contrast is more critical to peripheral signal detection than absolute stimulus luminance, particularly under hypoxia exposure.

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EFFECTS OF HYPOMIA ON PERIPHERAL VISUAL RESPONSE TO DIM STIMULI

by

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Several studies (Kobrick, 1965, 1971, 1972; Kobrick and Appleton, 1971; Kobrick and Dusek, 1970; Kobrick and Sutton, 1970) have shown that visual response time to flash stimuli increases in direct relation to the degree of peripheral placement of the stimulus. Furthermore, these findings have shown without exception that impairments are much greater for stimulus locations along the near-vertical axes of the visual field as compared to those for the horizontal axis. These findings have been corroborated by Haines and Gilliland (1973), who in addition have applied their data to make recommendations for design of improved aircraft instrument displays and cockpit window profiles, taking into account these inherent visual field limitations. Kobrick's findings have also shown that hypoxia produced by reduced inspired oxygen results in alterations of visual response in direct relation to the degree of hypoxic severity, but with a similar distribution of impairment to that found for normal sea level conditions. Haines (1973, 1973a) has reported similar impairment distributions for influences of acceleration, prolonged bed rest, and head tilt factors. Most recently, Kobrick (1974) found that the same impairment pattern of response to peripheral stimuli was generated under a task load requiring rapid, sustained performance for periods up to four hours, and that hypoxia had relatively less effect upon such performance

than it had upon task situations in which stimuli were infrequent in occurrence. In effect, the more frequent stimulation rate apparently acted to maintain attention, thus sustaining performance.

Another factor which could be expected to importantly influence a signal-detection task such as this is stimulus visibility, as determined by stimulus-background luminance contrast. However, such variables were not manipulated in the above research since they were not the primary interest, and because their inclusion with the other variables involved would have created unwieldy experimental designs. Instead, stimulus contrast was fixed at a level of moderately high visibility under daylight ambient lighting, a combination which was felt to be typical of many commonplace viewing situations. Even so, some estimate of the changes in peripheral visual response which could occur during other than the mid-viewing conditions used above would still be useful. Accordingly, the present study was conducted using the previous task, but in which dim stimuli distributed throughout the visual field were viewed against a dark background, and response time was measured during exposure to several levels of hypoxia.

Method

Subjects

Nine healthy male soldier volunteers, ages 18-25 years, were studied after screening for normal visual acuity (20/20 Snellen, uncorrected), normal peripheral vision, and for physical disabilities

which might be aggravated by hypoxia.

Apparatus and Procedure

The stimulus configuration of the experimental task was the same as used before by Kobrick in previous work, to afford the possibility of later comparisons. In brief, this consisted of 48 1/2-inch diameter white aircraft panel lights arranged in a hemispherically-shaped display about the visual field along 12 radial axes equally spaced at an angular separation of 30 degrees. Each axis contained four lights displaced 12°, 38°, 64°, and 90° angular subtense from the centerpoint of the display. The lights were flashed at 10-second intervals and in random order of locations, except that no light was repeated until all 48 had occurred. Thus, the stimuli were presented as complete 48-light series of different random orders but requiring the same length of time to complete. The subject's task was to orient to the center of the display and to press a switch held in the preferred hand as quickly as possible whenever a light was detected. The apparatus was situated inside a hypobaric chamber, and the experimental procedure exactly duplicated that used by Kobrick in previous work (1974), except for the stimulus luminances and the ambient lighting level. In the present study, the chamber was completely darkened, and each subject was pre-tested in the experimental apparatus to

determine the threshold luminance at which he could detect all of the most peripheral stimuli (90°) when flashed. Thereafter, all experimental runs were conducted under total darkness with all 48 stimulus lights set at the peripheral threshold value established for each individual subject; i.e., the luminance of all lights for each subject was set at the value he could just detect in the periphery. In actuality, the threshold values among the subjects were quite comparable (approximately 1 foot-lambert \pm 5%). The testing procedure was also the same as used previously, and consisted of initial training followed by identical 3 1/4-hour sessions at each of four hypobarically simulated elevations (0, 13,000, 15,000, 17,000 feet; or, 21%, 12.8%, 11.8%, 10.9% O_2 , respectively). Each session consisted of three work periods separated by 10-minute rests given at one-hour intervals, and contained a total of 22 stimulus series grouped into two units of eight series and one of six series. Sessions were administered to each subject in different counterbalanced orders, and were separated by one-week recovery intervals.

Results and Discussion

Data analysis followed the same form as used previously by Kobrick, and was based upon a unit score defined as the response time (RT) intervening between the onset of each stimulus light and the closure of

the subjects' switch. A log transform of these scores was first performed to normalize an apparently inherent skewness of their distribution, which had been observed in all previous data obtained on this task. All subsequent analyses were conducted using the normalized scores. A treatment x subjects analysis of variance was first performed on the total data to identify the principal treatment effects and interactions. Since one overall computer analysis could not handle the total data contained in the 22 stimulus series, the analysis was performed on stimulus series 2, 4, 6, 10, 14, 18, and 22 only. These series were selected in the previous study for the same reason, and since they proved to be quite representative of the total data trend, were used again in the same fashion. A summary of the analysis of variance is given in Table I, in which the F values were calculated with respect to their associated subject mean square interactions.

Table I about here

It can be seen that the experimental main effects of hypoxia (H), axis inclination (A), degree of peripheral stimulus location (P), and task duration as reflected by successive stimulus test samples (T), were all highly significant ($P < .001$), as in previous work utilizing this task. The results demonstrate once again the impairing effects of hypoxia on peripheral visual response.

However, in the previous study in which the same design was used, the simple interactions $H \times A$ and $H \times P$ were significant, whereas those in the present study were not. Conversely, the previous $H \times T$ interaction was not significant, but was significant in the present study. One would suspect from this that the effects of continued hypoxia were more pronounced than previously, yet in a more generalized fashion than could be identified by the interactions involving stimulus position. Even so, on the basis of the highly significant $A \times T$ interaction, one should expect to find a clustering of the impairment over the total exposure in certain zones of the visual field rather than in others. This clustering was, in fact, a typical finding in all of the author's previous work using this task. As an incidental observation, the highly significant $A \times P$ interaction verifies the ability of this analysis to reflect the relationship between the two factors which determine stimulus location.

In order to show the pattern of response impairment due to the separate hypoxia conditions over the total course of exposure, group means of the log RT's were obtained separately for each stimulus series involved in the analysis of variance. Two sets of group means were obtained on the same data, one set consisting of the RT's for each of the four peripheral stimulus locations averaged across all axis inclinations, the other set comprising the RT's for each of the 12 axis inclinations averaged across all four stimulus locations. This format is the same as that used in the previous study, and represents the present data in two ways; i.e., the effects of hypoxia on stimulus

peripheralization, and for different zones of the visual field. The group mean data were then plotted graphically as two sets of curves. Since presentation of all 14 of the resulting graphs in this paper would be prohibitive, only those for series 2, 10, and 22 are shown (see Figures 1-6) because these series effectively represent the overall trend of the present data as well as the peak impairment.

Figures 1-6 about here

The degree of impairment produced was directly related to the hypoxic severity involved and could be seen clearly in all of the graphs, especially the ones presented here. This amounted to very early-occurring moderate increases in RT's by 8-16 minutes of exposure (Figures 1 and 4), reaching maximum by 82-90 minutes (Figures 2 and 5), and diminishing thereafter to a level of performance which by 188-196 minutes (Figures 4 and 6) was approaching that at the beginning of exposure. Thus, although the trend of performance is similar to that observed before, the peak reduction took much longer to develop in the present study (82-90 minutes) than previously (24-32 minutes), a sizable difference of 58 minutes. Furthermore, the magnitudes of impairments at the various hypoxia levels were relatively smaller in the present study than in the previous one. Although these differences could be due to an overall difference in susceptibility to hypoxia between the two subject groups, this seems rather unlikely since they were all healthy young men and randomly drawn from Army populations in both studies. A more plausible explanation would seem to lie

in differences in stimulus characteristics, the only major variation between the two studies in an otherwise identical experimental design. Considering the stimuli used in both studies, a bright stimulus seen against a fairly bright background could in fact be functionally less visible than a much dimmer stimulus viewed against a dark background, because of the contrast advantage inherent in the latter. Since the subjects performed in the dark for well over 3 hours to complete the procedure, mesopic viewing sensitivity must have been achieved and maintained for most of the task; thus, any sensitivity bias should have been toward retinal rod than toward cone responding. Recognizing that the retinal periphery is more rod-populated, that the mesopic nature of the task biased the performance more toward rod receptor activity, and that all stimuli were set at a level visible in the periphery to begin with, it would seem safe to assume that the present task configuration was more functionally visible than that used in previous work. This could explain why the effects of hypoxia took longer to develop and were not as severe, since the stimuli were probably easier to detect under the test conditions.

Nonetheless, hypoxia did have its effect, producing decrements similar to those seen previously, even though they took longer to develop and did not reach the levels observed in former work. Considering all of the work done thus far using this task to study the effects of hypoxia on peripheral visual response, it is clear that the visual periphery, particularly about the medial axis, is markedly

vulnerable to the effects of hypoxic exposure. These findings are directly consistent with the severity of exposure, and from the present data are consistent with the contrast relationships of the stimulus surround of the viewing situation. Curiously enough, despite the general finding that brightness sensitivity and dark adaptation are particularly affected by anoxia (hypoxia) (Hecht, et al. (1946); McFarland and Halperin (1940)), it would appear that responding throughout the visual field under hypoxia can be greater for a dim stimulus with good contrast than for other brighter stimuli at higher ambient illumination levels if the latter are at poorer contrast with the surround. Thus, assessment of the effects of hypoxia on visual response must take account not simply of the virtual stimulus luminance, but rather of the functional visibility of the stimulus-background contrast relationships, assuming the stimulus to be above threshold value in the visual periphery.

Summary

Response times (RT's) of 9 Ss were obtained for detection of 48 flash stimuli distributed throughout the visual field during 3-1/4 hour exposures to each of 4 hypoxia conditions (0, 13,000, 15,000, 17,000 feet equivalent elevation). The luminance of all stimuli were set in common at the detection threshold value for the visual periphery. RT's were impaired in direct relation to hypoxic exposure severity, the peak impairments occurring within 90 minutes followed by gradual recovery. Since the present results showed less impairment

than previous data for brighter stimuli using the same task, it is concluded that stimulus contrast is more critical to peripheral signal detection than absolute stimulus luminance, particularly under hypoxic exposure.

References

- Haines, R. F. Effect of prolonged bedrest and $+G_z$ acceleration upon peripheral visual response time. *Aerospace Medicine*, 1973, 44, 425-432.
- Haines, R. F. Effect of passive 70° head-up tilt upon peripheral visual response time. NASA Technical Report, Ames Research Center, Moffett Field, CA, 1973.
- Haines, R. F., and Gilliland, K. Response time in the full visual field. *Journal of Applied Psychology*, 1973, 58, 289-295.
- Hecht, S., Hendley, C. D., Frank, S. R., and Haig, C. Anoxia and brightness discrimination. *Journal of General Physiology*, 1946, 29, 335-351.
- Kobrick, J. L. Effects of physical location of visual stimuli on intentional response time. *Journal of Engineering Psychology*, 1965, 1, 1-8.
- Kobrick, J. L. Effects of hypoxia on response time to peripheral visual signals. In The perception and application of flashing lights. Hilger, 1971, 323-335.
- Kobrick, J. L. Effects of hypoxia on voluntary response time to peripheral stimuli during central target monitoring. *Ergonomics*, 1972, 15, 147-156.
- Kobrick, J. L. Effects of hypoxia on peripheral visual response to rapid sustained stimulation. *Journal of Applied Physiology*, 1974, 37, 75-79.
- Kobrick, J. L., and Appleton, B. Effects of extended hypoxia on visual performance and retinal vascular state. *Journal of Applied Physiology*, 1971, 31, 357-362.

Kobrick, J. L., and Dusek, E. R. Effects of hypoxia on voluntary response time to peripherally located visual stimuli. *Journal of Applied Physiology*, 1970, 29, 444-448.

Kobrick, J. L., and Sutton, W. R. Device for measuring voluntary response time to peripherally placed stimuli. *Perceptual and Motor Skills*, 1970, 30, 255-258.

McFarland, R. A., and Halperin, M. H. The relation between foveal visual acuity and illumination under reduced oxygen tension. *Journal of General Physiology*, 1940, 23, 613-630.

TABLE I
SUMMARY OF ANALYSIS OF VARIANCE OF HYPOXIA, STIMULUS POSITION,
AND TASK DURATION EFFECTS ON RESPONSE TIME

| Source | df | Mean Square | F | P |
|----------------------------------|-----|-------------|-------|------|
| Hypoxia (H) | 3 | 19.73 | 8.77 | .001 |
| Axis inclination (A) | 11 | 3.37 | 37.45 | .001 |
| Peripheral stimulus location (P) | 3 | 39.41 | 70.38 | .001 |
| Stimulus test sample (T) | 6 | 2.79 | 4.50 | .001 |
| Subjects (S) | 8 | 5.44 | | |
| H x A | 33 | 0.10 | 1.25 | NS |
| H x P | 9 | 0.12 | 1.00 | NS |
| H x T | 18 | 1.03 | 1.67 | .05 |
| A x P | 33 | 5.54 | 61.56 | .001 |
| A x T | 66 | 0.07 | 1.75 | .001 |
| P x T | 18 | 0.03 | 0.60 | NS |
| H x A x P | 99 | 0.06 | 0.86 | NS |
| H x A x T | 198 | 0.04 | 1.00 | NS |
| H x P x T | 54 | 0.06 | 1.50 | NS |
| A x P x T | 198 | 0.07 | 1.75 | .001 |
| H x A x P x T | 594 | 0.04 | 1.00 | NS |
| H x S | 24 | 2.25 | | |
| A x S | 88 | 0.09 | | |
| P x S | 24 | 0.56 | | |
| T x S | 48 | 0.62 | | |

TABLE I (cont'd.)

| Source | df | Mean Square | F | P |
|---------------|-------|----------------|---|---|
| H x A x S | 264 | 0.08 | | |
| H x P x S | 72 | 0.12 | | |
| H x T x S | 144 | 0.62 | | |
| A x P x S | 264 | 0.09 | | |
| A x T x S | 528 | 0.04 | | |
| P x T x S | 144 | 0.05 | | |
| H x A x P x S | 792 | 0.07 | | |
| H x A x T x S | 1584 | 0.04 | | |
| H x P x T x S | 432 | 0.04 | | |
| A x P x T x S | 1584 | 0.04 | | |
| Residual | 4752 | 0.04 | | |
| Total | 12095 | | | |

Figure Captions

- Figure 1. Group mean response time (RT) at each peripheral stimulus position under each level of hypoxia during 8-16 minutes of exposure (stimulus series 2).
- Figure 2. Group mean response time (RT) at each peripheral stimulus position under each level of hypoxia during 82-90 minutes of exposure (stimulus series 10).
- Figure 3. Group mean response time (RT) at each peripheral stimulus position under each level of hypoxia during 188-196 minutes of exposure (stimulus series 22).
- Figure 4. Group mean response time (RT) at each axis inclination under each level of hypoxia during 8-16 minute of exposure (stimulus series 2).
- Figure 5. Group mean response time (RT) at each axis inclination under each level of hypoxia during 82-90 minutes of exposure (stimulus series 10).
- Figure 6. Group mean response time (RT) at each axis inclination under each level of hypoxia during 188-196 minutes of exposure (stimulus series 22).

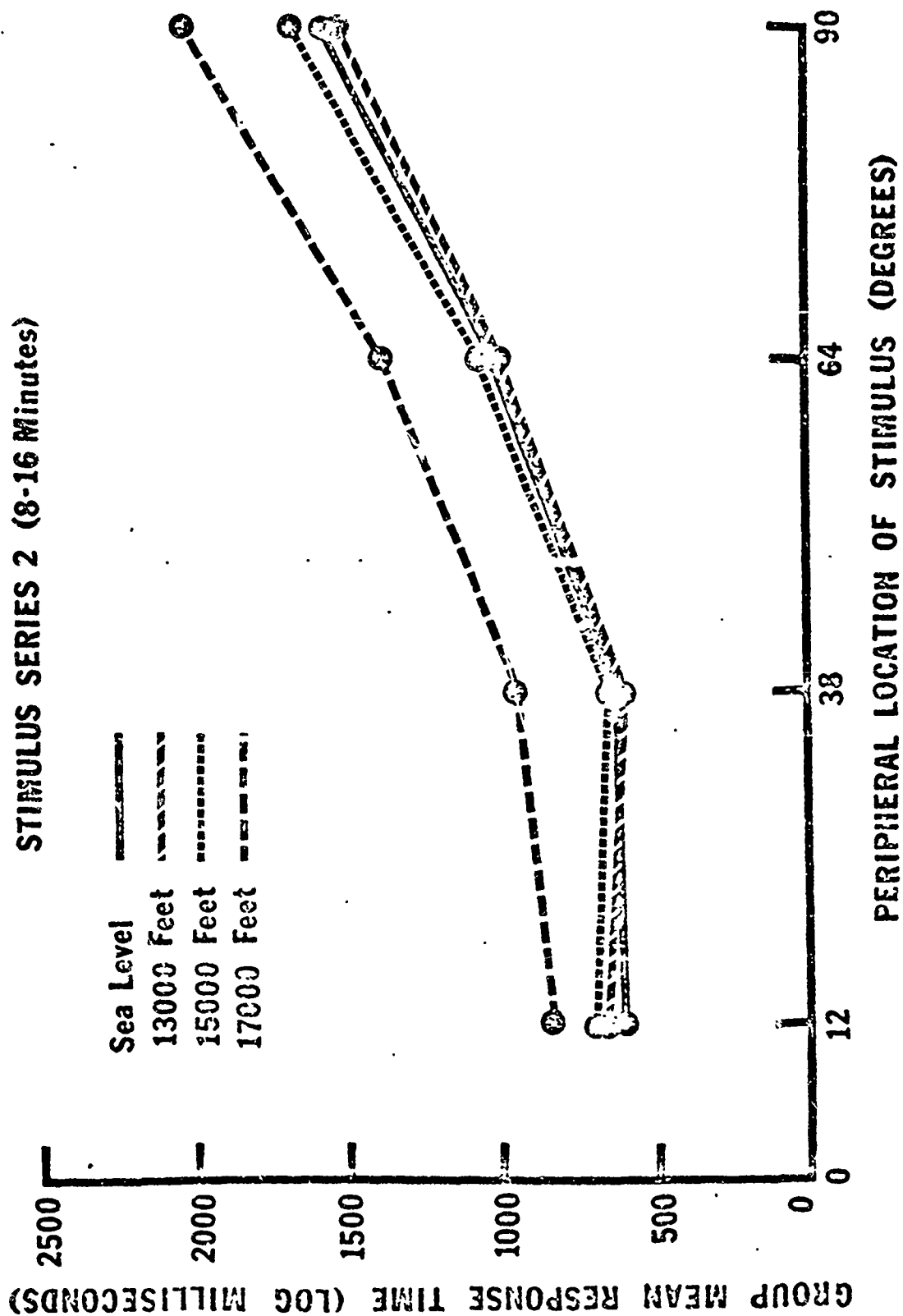


FIGURE 1.

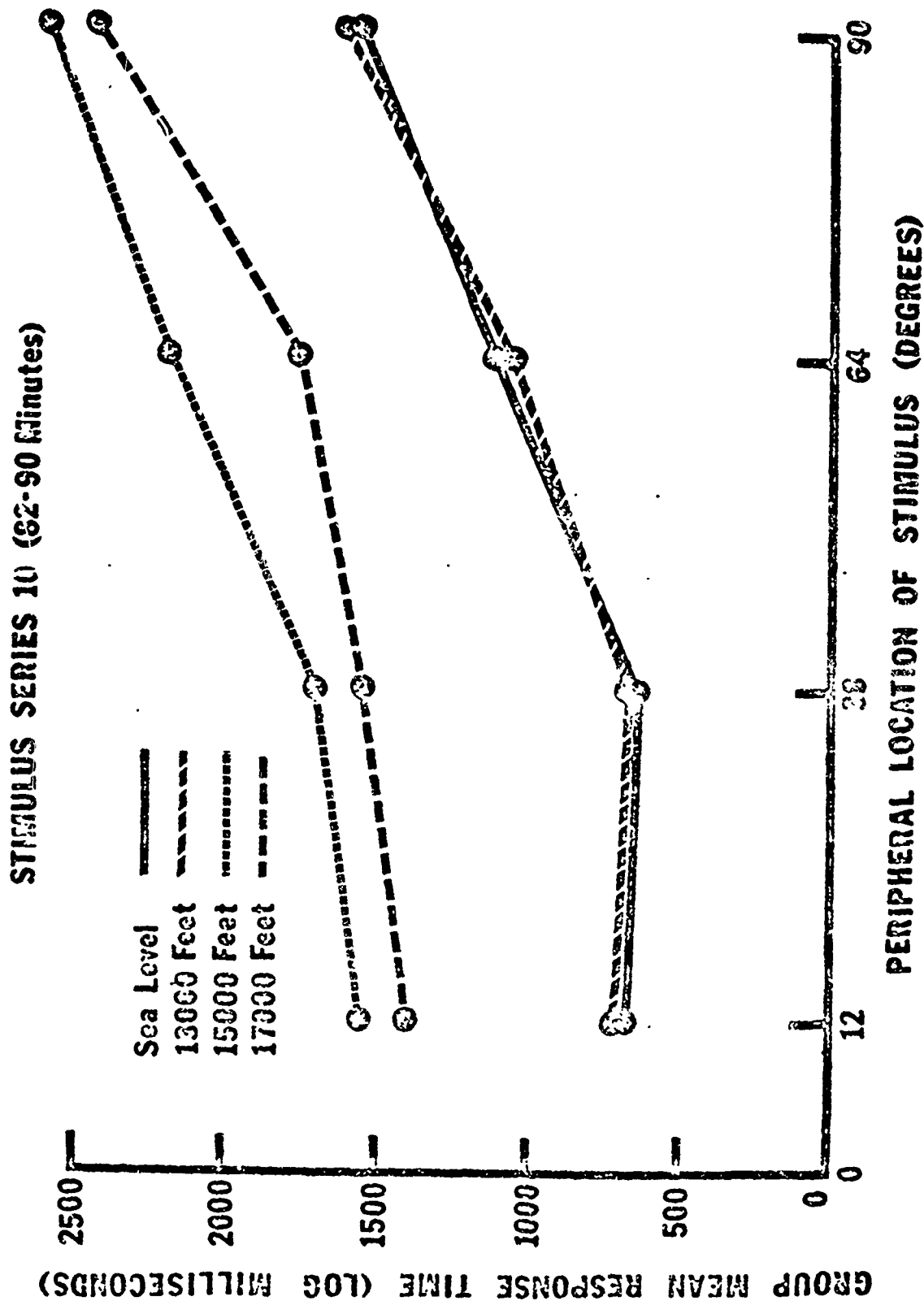


FIGURE 2

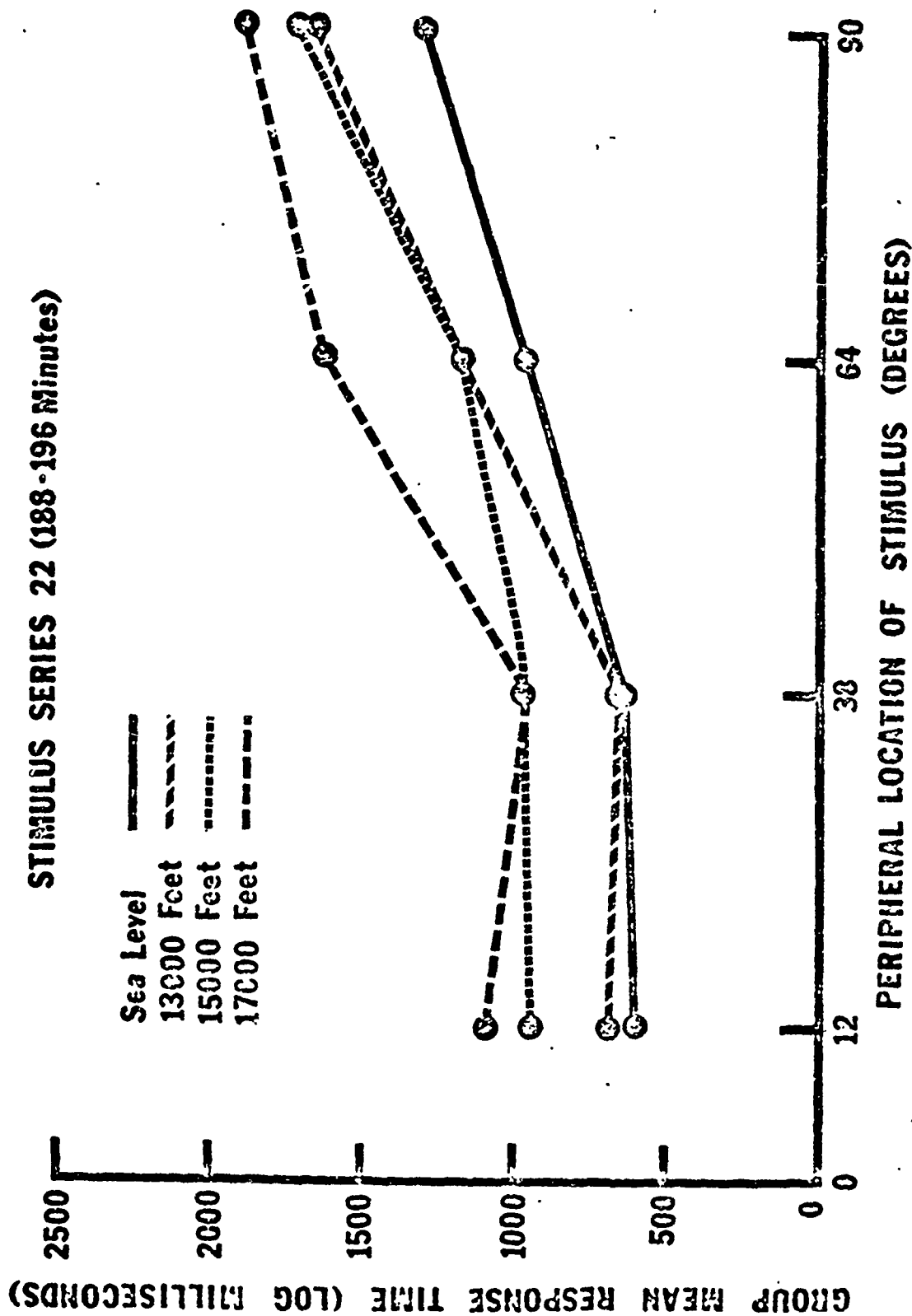


FIGURE 3

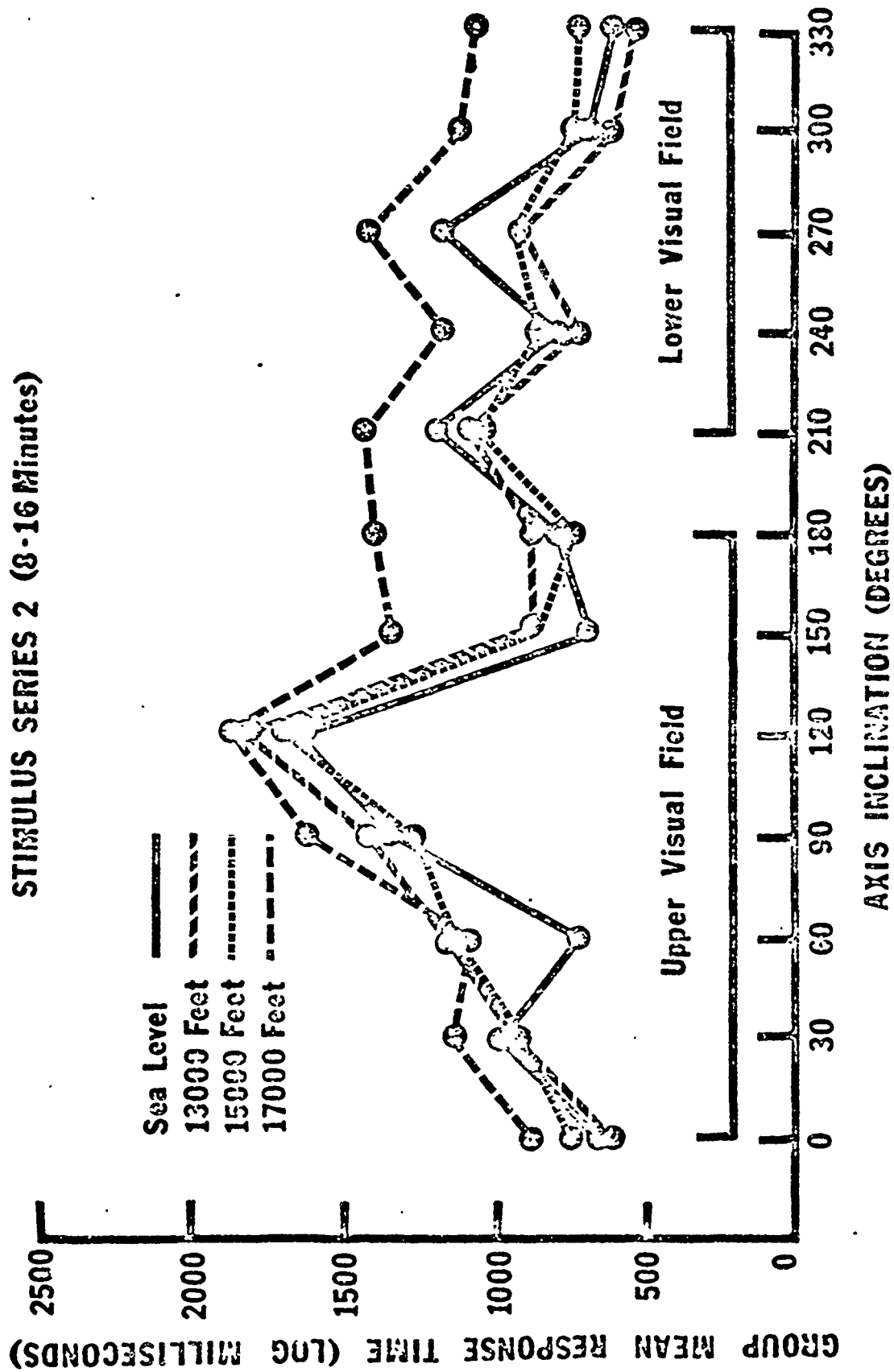
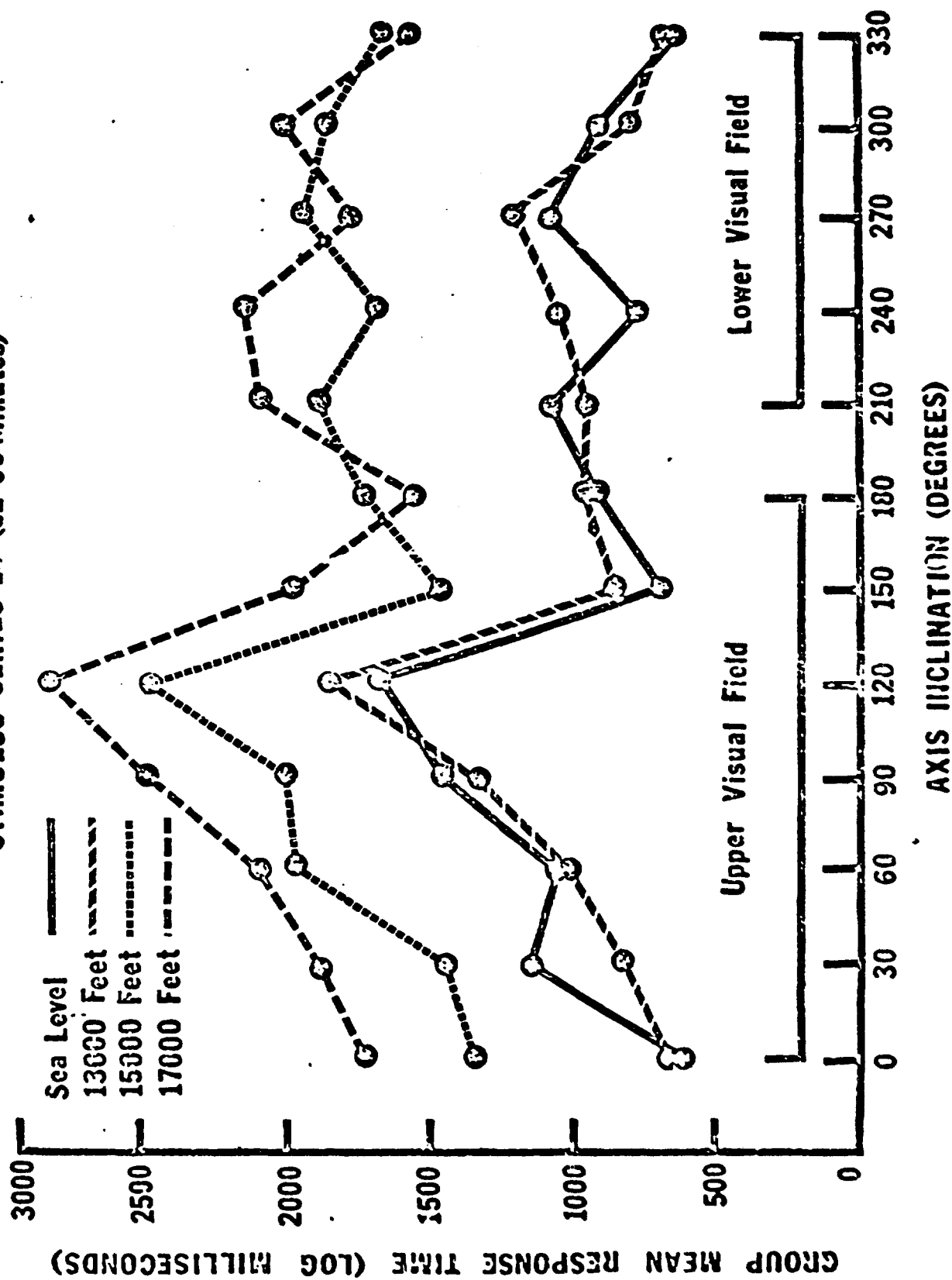


FIGURE 4

STIMULUS SERIES 17 (82-90 Minutes)



STIMULUS SERIES 22 (188-196 Minutes)

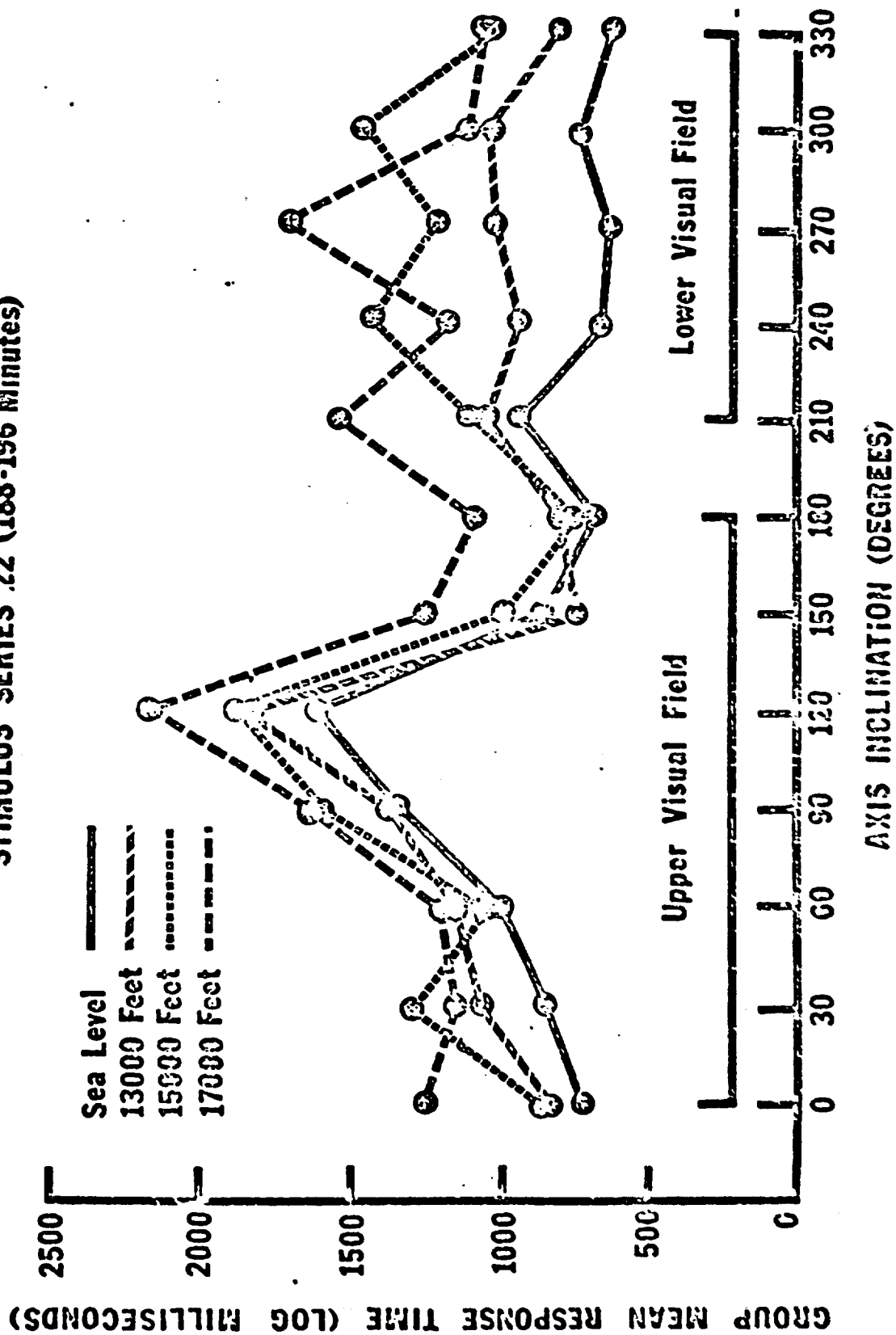


FIGURE 6

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